

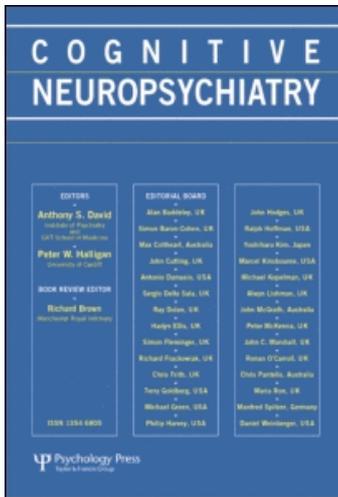
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Novel insights into false recollection: A model of déjà vécu

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The thesis of this paper is that déjà experiences can be separated into two forms: déjà vu, arising from the erroneous sensation of familiarity, and déjà vécu, arising from the erroneous sensation of recollection. We summarise a series of cases for whom déjà vécu is experienced frequently and for extended periods, and seek to differentiate their experiences from “healthy” déjà experiences by nonbrain-damaged participants. In reviewing our cases, we stress two novel ideas: that déjà vécu in these cases is delusion-like; and that these cases experience déjà vécu for stimuli that are especially novel or unusual. Here we present a novel cognitive neuroscientific hypothesis of déjà vécu. This hypothesis assumes that the signal of retrieval from memory is neurally dissociable from the contents of retrieval. We suggest that a region downstream of the hippocampus signals “recollection” by detecting the timing of firing in hippocampal output neurons relative to the theta oscillation. Disruptions to this “temporal coding” mechanism result in false signals of recollection which may occur without actual retrieval and which, ironically, may arise particularly during situations of contextual novelty.

Keywords: Déjà vu; Dementia; Hippocampus; Theta.

INTRODUCTION

“From the point of view of episodic memory . . . each event a person experiences is always unique in the sense that in all of his previous autobiographical history there has not been another experience exactly like the present one.” (Tulving, 1972, p. 399)

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Following an influential review on the topic (Brown, 2003), there has recently been an increase in interest amongst cognitive psychologists in the *déjà vu* phenomenon and associated states. Researchers have converged on a definition of *déjà vu* that emphasises the (erroneous) sensation of familiarity, and the subjective nature of the state. In healthy people, it is hypothesised that the state arises temporarily as a result of an erroneous sensation of familiarity. Indeed, using a fairly standard paradigm for inducing familiarity (the mere exposure paradigm) it has been possible to induce *déjà vu*-like sensations in the laboratory amongst undergraduates (see Brown & Marsh, 2008).

However, a major theoretical viewpoint within contemporary memory research is that memory retrieval can be based on two distinct processes or states: remembering and knowing (Tulving, 1985). In brief, these two states map on to two distinct processes. Remembering is an effortful recollective process associated with retrieval from episodic memory (corresponding to auto-noetic consciousness) and knowing, or familiarity, is a more automatic process, associated with retrieval from semantic memory (corresponding to noetic consciousness; see also Gardiner, Ramponi, & Richardson-Klavehn, 1998).

Remembering and knowing have been found to be associated with distinct and dissociable neural correlates (e.g., Henson, Rugg, Shallice, Josephs, & Dolan, 1999, and see later). According to an emergent view (see Conway, 2005), memory is accompanied by experiential states that help the memory-user to infer the quality and veracity of the retrieved information—retrieval from episodic memory can be interpreted as such due to the sensation of remembering (this may be particularly important in differentiating remembering from imagining, see Addis, Wong, & Shacter, 2007), whereas a feeling of familiarity can be interpreted as resulting from a previous encounter in the absence of such evocative recall. In short, our view is that two distinct processes are also involved in abnormal memory experiences; as well as erroneous sensations of familiarity, there will be erroneous sensations of remembering.

We also propose that all **sensations associated with retrieval can become dissociated from the act of retrieval itself. In these situations, there is a clash between the sensation of retrieval and the actual contents of retrieval itself.** Thus, the *déjà vu* experient momentarily feels like she has previously visited a never-visited location in the absence of being able to retrieve memories of any such visit (and even whilst bringing to mind the knowledge that she has never been to the location). We argue that it is likewise possible to have erroneous sensations of remembering. Our argument is chiefly based on a series of cases. Moulin and colleagues (Moulin, Conway, Thompson, James, & Jones, 2005; Moulin et al., 2006) describe patients with dementia who experience erroneous sensations of *remembering*. These patients (described in detail later) report that events have happened before, even though they are experiencing them for the first time. These cases also show *recollective*

secondary confabulation—the patients confabulate contextual and circumstantial material to justify their overwhelming sensations of events having happened before. We describe this recollective memory error as *déjà vécu* in order to differentiate it from disorders of familiarity, which can also involve clear memory deficits, and presentations just as striking as those displayed by our patients. Indeed, although erroneous familiarity is often experienced in the healthy population, it can also manifest in ways that warrant clinical attention. For instance, Ward et al. (1999) describe a set of patients who feel, on the basis of familiarity, that everyone they meet is “famous”.

Therefore, in this paper we aim to extend our theory of *déjà vécu* as a disorder of remembering, which is qualitatively different to, rather than quantitatively different to (in terms of degree or the requirement for clinical intervention) disorders of familiarity. In doing so, we posit a possible mechanism for its formation, attempt to differentiate it from other manifestations of the *déjà vu* experience, and offer a more specific means by which to identify such cases: inappropriate sensations of remembering for *novel stimuli*.

DÉJÀ VU AND DÉJÀ VÉCU

The consequence of reporting these clinical cases of *déjà vécu* (and in particular their coverage in the mainstream media), is that the term has come to be described merely as a clinical manifestation of the *déjà vu* experience. This confusion is further compounded by the fact that clinicians and carers frequently and spontaneously describe their cases and family members as having *déjà vu*, even though there are marked differences between the clinical presentation of *déjà vécu* and “healthy” instances of *déjà vu*. We suggest that healthy people do experience a nonpathological form of *déjà vécu*. (For descriptions of *déjà vu*- and *déjà vécu*-like sensations induced in healthy participants see O’Connor, Barnier, & Cox, 2008.) This is possible in the same way that we may experience *déjà vu* for a fleeting instant when meeting a stranger in a supermarket but not have a full-blown neuropsychological deficit whereby we believe everyone to be famous (Ward et al., 1999). However, the prevalence of nonpathological *déjà vécu* in the healthy population is unknown.

In using the term *déjà vécu*, we deliberately borrowed from Funkhouser’s (1996) fractionation of *déjà vu* into three separate experiences: *déjà vécu*, *déjà senti*, and *déjà visité*. Funkhouser’s definition of the *déjà vu* states is drawn from both scientific works and literature. Notably, to describe *déjà vécu* he uses an oft-cited passage from Dickens’ *David Copperfield*:

“We have all some experience of a feeling, that comes over us occasionally, of what we are saying and doing having been said and done before, in a remote time—of our

having been surrounded, dim ages ago, by the same faces, objects, and circumstances—of our knowing perfectly what will be said next, as if we suddenly *remember* it!” (Dickens, 1850, p. 236, emphasis added)

This description stresses contextual details and associated feelings and thoughts. As such, it parallels recollection, where in remembering an event we are able to draw up contextual information, and the specifics of a previously experienced event (e.g., Conway, 2005). In contrast, Funkhouser (1996) describes *déjà senti* as a more nebulous sensation, and differentiates it from *déjà vécu* thus as being “primarily or even exclusively a mental happening” with no precognitive aspects. To illustrate this sensation he draws on Hughlings Jackson’s account of temporal lobe epilepsy:

“[W]hat is occupying the attention is what has occupied it before, and indeed has been *familiar*, but has been for a time forgotten, and now is recovered with a slight sense of satisfaction as if it had been sought for.” (Hughlings Jackson, 1888, p. 202, emphasis added)

This passage describes a mental event which, unlike recollection, is incomplete, with an absence of recollective detail, and the “reliving” of a situation. Funkhouser’s final subdivision is *déjà visité*. This is a classification of *déjà vu* based on it occurring in particular locations—“an experience in which a person visits a new locality and nevertheless feels it to be familiar” (Funkhouser, 1996). We do not see this as a separate experiential state but a description of a *déjà* experience triggered by a particular cue or event. We actually have accounts of both *déjà vu* and *déjà vécu* cued by location, so suggest that this subdivision does little to help us understand the underlying memory processes at fault in the experience, although it may be important in classifying the experience in other ways not attempted here.

We emphasise the difference between the first two categories, although we chose not to use the term “*déjà senti*”, even though it appears to resonate with our characterisation of false recognition on the basis of familiarity. This is largely because, despite Funkhouser’s (1996) observant classification of the varieties of *déjà vu*, the literature has converged on a description of *déjà vu* that emphasises the experience of *familiarity*—“... any subjectively inappropriate impression of familiarity of a present experience with an undefined past” (Neppe, 1983, p. 3). Neppe’s definition of *déjà vu* points to another issue we see as critical. Twice in his definition, he refers to the experient’s sensation of the experience. First, the experience is “subjective” and second, his definition clarifies that the experience must be combined with an “undefined” past. That is, if someone finds something familiar, and can identify why or where from, it is not *déjà vu*. This introduces the second area where we think the literature is lacking precision—*awareness*. Nonpathological *déjà*

experiences involve self-awareness. We are not seduced by our “healthy” déjà vu experiences, no matter how strong—we will report a feeling of familiarity, but we will not be able to produce a past experience in order to justify it, and so we will acknowledge that our sense of familiarity is erroneous. Healthy individuals will not act on the basis of their experiences of déjà states, no matter how frequent or persistent.

Our patients, in contrast, are characterised by a persistent and overwhelming occurrence of déjà experiences, which they act upon and about which they mostly lack awareness. Clinical reports of persistent and debilitating forms of the déjà experience have been made of those with dementia, temporal lobe epilepsy, migraine, and schizophrenia (Kalra, Chancellor, & Zeman, 2007; Moulin et al., 2005; Neppe, 1983; Sacks, 1970), but it is not clear whether these experiences are déjà vu or déjà vécu, since the case descriptions and neuropsychological examination often provide insufficient detail to make this distinction.

A further confusion in the use of déjà vécu and déjà vu may be historical. Our interpretation of one of the early neuropsychological cases that used the then new term “déjà vu” is actually more a case of déjà vécu. **Arnaud (1896) describes Louis, a 34-year-old male who had suffered anterograde and retrograde amnesia as a result of contracting cerebral malaria. Following his recovery, Louis reported recognising everything he encountered. Arnaud tested his patient’s memory by exposing him to a number of events and documenting Louis’ experiences of these events. One of these events was Pasteur’s state funeral, a highly distinctive event attended by thousands. Arnaud reported that Louis described the funeral as an event he recognised, demonstrating that his erroneous recognition was experienced not only for mundane events, but also for highly distinctive and novel events, a theme we will pick up again later in this paper.**

CASE REPORTS OF DÉJÀ VÉCU

Moulin et al. (2005) initially reported the cases of two patients, AKP and MA, who experienced déjà vécu associated with dementia. AKP was an 80-year-old man who initially presented to his family doctor with memory problems and frequent sensations, which were described as déjà vu. MA was a 70-year-old woman who presented to her doctor, convinced that things had happened before, as evidenced by her memory for them happening as they happened. AKP and MA’s erroneous sensations of memory were so compelling that the patients were unwilling to engage in activities such as watching television and reading the newspaper, as they felt they had already carried them out. As mentioned previously, healthy déjà experiences do not lead to behavioural change—one is able to challenge and resist the sensation

that one has encountered something before. Conversely, déjà vécu patients act on their beliefs. See Table 1 for other examples of déjà vécu patients' experiences.

TABLE 1
Accounts of déjà vécu patients' experiences as reported by correspondence with carers

<i>Age</i>	<i>Diagnosis</i>	<i>Description</i>
74	Memory difficulties	"Chronic déjà vu. I can't find any explanation, or correlation with any of her physical ailments ... it gets worse during stress ... has become a chronic (multiple times a day). It is bothering to her and worrisome to me. It is triggered by both novel events and mundane, common occurrences, but much more prevalent with change of environment."
85	Early dementia	"She doesn't read or watch TV because "she's seen or read them all before". She even told me that CBC must be on strike because they are running re-runs ... we were out for a drive and were about 20 km from home. We passed a couple walking along the side of the road and mom said she had seen them earlier that morning when she was out for her walk around the block. I tried to explain that it was doubtful that it could be the same couple but she insisted that she could describe the coat the lady was wearing and proceeded to do so."
—	Stroke	"One of the most striking features of her brain injury was this newfound sense of having seen every single show on television before, years ago, when she was younger. This was, ironically I suppose, coupled with an inability to remember what had happened at the beginning of the show by the time the program ended. She also developed a tendency to make up memories from her past that had never actually happened (childhood events and the like) and recounts them very detailedly, never realising that they are in fact false."
78	Unknown	"My parents decided to not let the déjà vu's interfere with their winter vacation and went to Florida. My Dad reported that the déjà vu's were stronger and more frequent. Museums, restaurants, beaches, stores and people never visited, were all seen/felt/done before. They were worse than when at home. ... At the flower show a week back, we were standing there, looking at a display and she said 'oh I saw that before, last night on TV. I think' then, she saw a lady pass us and said, 'you've seen her, she's on TV I know I've seen her before'."
86	Unknown	"In the last two years of her life (she died at 88) I would have to say that she essentially lived in almost a constant state of déjà vécu whenever she was removed from her narrowly defined daily routine (in an assisted living residence). Whenever she spent time with me, for example, there was continual reference to 'the trees have sure grown taller,' total strangers on the road had 'gotten fat and, look, he has a new dog,' and every stranger she met was assured that she knew them already. Newspapers had been read before, she had repairmen in to find out why the television kept showing programs she had seen before, newly published books had been read years before, etc."

In order to establish the recollective nature of *déjà vécu*, Moulin et al. (2005) administered a recollective experience-based recognition task (Table 2) to AKP and MA. Using this task, they were able to ascertain a typical response pattern in those with *déjà vécu*. When they compared the *déjà vécu* patients' responses to those of age-matched controls, the *déjà vécu* patient responses were characterised by a greater number of false positives (FPs, which occurs when a new item is incorrectly identified as being seen before) and by a higher proportion of those false positive as being "remember" responses (Table 3). Thus, according to these measures of recollective experience, AKP and MA had a far greater tendency to "remember" items they had not been shown at study. Moreover, they tended to confabulate contextual details associated with items they had not previously encountered (i.e., they reported experiences and thoughts from the nonexistent study phases for nonpresented items).

In fact, this recollective secondary confabulation seems to be a critical feature of this condition. (We refer readers to Turner and Coltheart and Langdon and Bayne, both this issue 2010, for discussion of secondary, or post hoc, confabulations. Hereafter, we will simply use the term "recollective confabulation".) Since recollection is associated with the retrieval of

TABLE 2
The recollective experience procedure

<i>Recollective experience memory tasks (Gardiner, Ramponi, & Richardson-Klavehn, 1998; Tulving, 1985) assess recognition memory by giving participants the opportunity to respond in the following ways to stimuli presented at test</i>	
Remember (R)	I recognise this word. I can remember aspects of my previous experience of encountering it before (e.g., what it made me think).
Familiar (F)	I recognise this word. I don't remember any aspects of previously encountering the word but I just know it was in the previous list.
Guess (G)	I think I recognise this word, but I'm guessing.
No recollection	I do not recognise this word. This is a word I didn't encounter before.

TABLE 3
Summary of results for AKP, from Experiment 2, Moulin et al. (2005)

Score (max. =20)	<i>Hits assigned to each subjective category</i>			<i>False positives assigned to each subjective category</i>		
	<i>Remember</i>	<i>Familiar</i>	<i>Guess</i>	<i>Remember</i>	<i>Familiar</i>	<i>Guess</i>
AKP (z-score)	15 (-0.78)	3 (1.93)	2 (3.34)	8 (14.55)	3 (3.73)	3 (5.67)
Controls (SD) (n = 7)	17.16 (2.75)	0.84 (1.12)	0.21 (0.54)	0.21 (0.54)	0.26 (0.73)	0.16 (0.50)

contextual information and experiences at study, it is perhaps unsurprising that participants who experience high levels of déjà vécu also generate high levels of incorrect contextual information for items which they have not actually encountered before. AKP made such reports spontaneously, for instance confabulating secret early-morning trips to the newsagents to read the newspaper as it was unloaded from the lorry, whilst his wife was asleep in bed. We think that these confabulatory justifications are similar to the justifications made by anosognosic patients with hemiparesis (see Fotopoulou, this issue 2010).

This tendency to confabulate was noted in Mr. K, reported by O'Connor (2007). Mr. K was a 92-year-old man who started experiencing memory difficulties in his 80s. Mr. K's déjà vécu became apparent to his son when they took a family holiday to France, a country Mr. K had never previously visited. His son reported an episode of recollective confabulation that is notable for the insight Mr. K had:

"When we travelled round France, my father saw a hospital and said, "I have been here before, I visited this hospital to see my friend." He then told the story of his previous visit to confirm his memory. When he was asked how this could be possible when he had never visited France before, my father said, "I know that I have never been here when I consider my personal history seriously and logically, but I still strongly feel that I have been here before." We thought he was over-tired so we took a two-day break from sightseeing. Unfortunately, when we resumed sightseeing his memory situation had not changed." (O'Connor, 2007, p. 214)

The dissociation between Mr. K's feelings of recollection and his awareness that he could "logically" not have been to the hospital in France illustrates the conflict caused by Mr. K's déjà vécu—it was only when challenged that Mr. K would concede that his sense of recollection could be false. However, such was the strength of his memory sensation and the associated confabulation that unless he was forced to consider the consistency of his memories with what he knew about his past experience, Mr. K would believe that his memories were accurate.

Persistent déjà vécu as delusion-like

Mr. K's partial awareness of his condition is somewhat typical of our cases. We suggest that our patients are broadly speaking anosognosic, but that they do have some insight into their difficulties. Tellingly, when AKP was challenged by his wife to remember the contents of the premiere of a TV show he believed he had watched before, AKP retorted: "How should I know, I have a memory problem!" (Moulin et al., 2005, p. 1364). Similarly, a new case, Mrs. M (Moulin & Conway, 2009) reported that although it felt

like everything in her life was repeating, it was often possible to just sit with her head in her hands in the kitchen, and work it all out, describing her experience as very confusing. AKP, in justifying his decision that he had previously encountered a word earlier (in fact he had not been presented with the word) reported that he was certain he had seen the word earlier, but could not remember the study phase in which he had been presented with it. His own description of his condition was that he did get confused about whether events had repeated or not, and yet, he still felt compelled to act on his memory feelings.

We suggest that it is the dimension of insight that sets apart a healthy déjà experience from a pathological one. Here we argue that the lack of insight means that our clinical cases of déjà vécu are delusion-like. During a healthy déjà experience, the experient is aware that he or she is experiencing erroneous familiarity or recollection and so does not accept or act on the sensation. During a pathological déjà experience, the experient does not seem to be aware that the sensation is erroneous, and therefore modifies his/her behaviour in accord with the sensation (e.g., withdraws from the “repeated” activity). However, not all cases are as straightforward as this. For instance, a feature of Mr. K’s déjà vécu was his acknowledgment of the possibility that his sensation of recognition could be erroneous. Not only did Mr. K acknowledge that his recognition could be erroneous, but he admitted that, “logically”, it probably was erroneous. When challenged, Mr. K could be made aware that his sensation of recognition was false.

Our déjà vécu patients share important features with clinical cases of delusions. The operationalisation of the DSM-IV (American Psychiatric Association, 1994) definition of delusion has been debated, but there is some consensus that delusions run counter to the beliefs of peers, are maintained despite overwhelming evidence to the contrary, and defy rational counter-argument. These same characteristics can be seen in déjà vécu. According to Langdon and Coltheart (2000), the interaction of two factors is necessary for the formation of a delusion. These are: a neuropsychological anomaly that alters perceptual or emotional processing and so disrupts the “perceived reality”; and damage to a system of belief evaluation that is responsible for the person’s failure to reject the erroneous belief which stems from the first neuropsychological anomaly. In a similar vein, déjà vécu could be seen as a dysfunction of the recognition systems coincident with damage to systems responsible for evaluating and correcting the information which stems from this dysfunction. Here we develop this idea in relation to memory systems, with the notion that frontal systems should normally be able to discount erroneous sensations emanating from memory processes occurring in the temporal lobe, but that in these cases of déjà vécu, this system is deficient.

In both healthy and pathological déjà experiences, dysfunctions of recognition may result in a combination of the feeling of recognition and

the awareness that this feeling is inconsistent with the knowledge and beliefs we hold about our previous experience. At this stage of the experience, healthy and pathological experiences could be said to result from similar neuropsychological anomalies. The point of divergence is in the system of belief evaluation. Whereas in healthy *déjà* experiences, the dysfunctional recognition is corrected in favour of the evaluation that is consistent with the rest of the experient's knowledge, in pathological *déjà* experiences, and particularly in our cases of *déjà vécu*, the dysfunction is not corrected but allowed to coexist alongside divergent knowledge and beliefs held about previous experience and the likelihood that the recognition being experienced is inappropriate. In healthy experiences, a unified experience is maintained, but in pathological *déjà vécu*, the absence of an appropriate evaluation of consistency results in a fragmented, inconsistent experience. The proposed evaluative dysfunction in *déjà vécu* can account for both recollective confabulation and the awareness that Mr. K demonstrated when asked to reconcile his recollective confabulations with his knowledge of his previous experience. He was comfortable with the use of the confabulated memory of visiting a friend in the hospital to justify his feelings of recognition in France. However, this confabulated recollection did not appear to have any consequences for the way in which Mr. K then evaluated everything else he knew about his own life, such as the fact that he had never previously visited France.

Novelty: On the triggers of *déjà vécu*

One of the more noteworthy features of AKP's presentation was that it seemed that it was more striking, less commonplace, distinct events that triggered more intense sensations of *déjà vécu*. On hearing of the death of a close friend, for instance, he claimed that he had already heard the news and he even refused to go to the funeral, claiming that he had already been. Similarly, patient MA produced *déjà vécu* for striking news events, such as the Bali bombing (Moulin et al., 2005), which she claimed to have already known about. In contrast, mundane and repetitive acts, such as acts of personal hygiene and eating meals, never elicited *déjà vécu* experiences. Table 1 shows examples of carers' reports of people with similar or identical symptoms—note how often this includes novelty, or at least the observation that this problem is magnified in new locations or nonroutine events. In our original presentation of AKP and MA, we operationalised this observation by examining false positives and recollective experience for high frequency (common) words (such as *mouse*) and low frequency (distinctive, rare) words (such as *polka*). Somewhat counterintuitively, **it was the low frequency words that were more likely to elicit false positives and recollective experience.** This

suggested that the *déjà vécu* experience was not made on the basis of familiarity, or confusing the source of a highly familiar word, but due to recollective processes specifically triggered by more novel, or striking events.

We argue that demonstrating such patterns of performance is critical when investigating a state as nebulous and subjective as *déjà vécu*. O'Connor, Moulin, and Cohen (2007) outline a series of considerations when examining subjective states such as *déjà vu*. These authors emphasise the need to show that subjective evaluations and states vary predictably according to objective qualities of stimuli, that evaluations are supported by verbal justification and that the experient can spontaneously draw parallels with naturally occurring day-to-day sensations. All of these were satisfied in AKP and our other cases. Our final consideration is that neuroscientific findings should seek to support the validity of the experience and the behavioural observations. For instance, in something as subjective as the perception of Rubin's vase, markedly different patterns of brain activation have been shown according to whether the participant sees it as two faces or a vase (Andrews, Schluppeck, Homfray, Matthews, & Blakemore, 2002). We have not performed neuroimaging or electrophysiological recording on any of our cases. This should be a priority in future research. Accordingly, we proceed to outline a testable neuroscientific hypothesis on how *déjà vécu*, as experienced in our cases, might arise.

Importantly, the strange experiences of AKP and MA are consistent with subsequent cases. For instance, Mrs. M (Moulin & Conway, 2009) who presented to us having complained to the BBC about the high levels of repeats on television, and also as having called a television repairman to fix her television, had the same pattern of memory errors. Mrs. M was unhappy watching new television programmes, in particular, the soap opera, "Neighbours", but was happy watching old films that she had already watched before. In the case of "Neighbours", she described her experience as frustrating to the point of tears, and as waiting for the programme to stop repeating. Ironically, whilst watching DVDs of old films, she said she could happily watch them again, as she had forgotten what had happened. We operationalised such a memory error by using a "reverse" Deese-Roediger-McDermott (DRM) task. In the DRM task (Roediger & McDermott, 1995), semantically related items presented at study converge on a nonpresented lure word (e.g., *thread*, *haystack*, *cotton*, and *pin* all converge on the word *needle*). Healthy participants are likely to report that they remember the word *needle*, even though it was never presented. With Mrs. M we presented similar lists of related words but with one critical *unrelated* lure. Following presentation of related concepts such as colours (red, green, blue, black), a healthy participant is very likely to incorrectly remember a related concept such as *yellow*, whereas they are very unlikely to report remembering an unrelated, distinct item such as *Christmas*. We found that Mrs. M made the

unlikely errors—even though all of the items presented at study were categorised, and she could report the title of that category, she was still liable to incorrectly recognise items that were strikingly different and not part of that category. Finally, in meeting déjà vécu cases in their own homes for the first time, it has always been our experience that they claim to have met us like this before. Interestingly, this error is not made on subsequent visits. Converging on this novelty view, several carers of patients with such déjà vécu have also reported similar situations in their e-mail correspondence (Table 1).

Deja vécu is reported with other aetiologies, for example, where a patient had received 5-hydroxytryptophan in combination with carbidopa, as treatment for palatal tremor (distressing, repetitive, and involuntary movements of the soft palate; Kalra et al., 2007). Her account also indicates that novelty is possibly a trigger:

“I . . . was a little freaked out when I watched TV as I felt I was watching repeats, although I knew I wasn't, as it was the news. I then got a phone call from my sister to tell me the kids were being sent home as there was a power cut at school. I asked her why she was telling me this again as she had told me this several days before. She asked me if I was okay and asked how she could have known that there was going to be a power cut.” (Kalra et al., 2007, p. 312)

One interpretation of our novelty observation is that novel situations and events do not preferentially elicit acute instances of déjà vécu, but rather render a largely chronic déjà vécu state more noticeable. Perhaps our patients are chronically experiencing inappropriate sensations of remembering, but it is only novelty that brings these sensations to the attention of the patients (and carers). That is, when the patients are cleaning their teeth they may feel that they recollect an event, but since this event is commonplace, this sensation of remembering is not so jarring. In contrast, perhaps when Mrs. M watches a film on TV that is actually new to her, the feeling of recollection she experiences is more obviously inconsistent with her stored knowledge, which ought to support the belief that the film has not been seen before: thus the sensation of her (false) recollection is brought forcibly to the forefront of her mind. Such an account does receive some support from the literature on healthy déjà vu experience; it appears that people who travel more report more déjà vu (see Brown, 2004, for a review). Travelling may present more opportunity to experience dissociations between memory sensations and knowledge of previous events. Consider this extreme example: If you did actually attend the same places repeatedly and never travelled elsewhere, you would always have a justification for your feeling of familiarity. In the same way, patently novel events may provide time-limited windows for our patients to experience feelings of recollection that are factually unjustified.

An alternative interpretation, in line with our current thinking is that novelty does genuinely preferentially elicit acute episodes of *déjà vécu* in these patients. For instance, and as noted previously, we are struck by the observation that when we first meet our patients, they say they have met us before, but do not say this on the second or third time we meet them. Accordingly, we propose a specific neuroscientific mechanism underlying *déjà vécu* that accounts for this ironic error, and we build a case for this novel account. The account is discussed later as the encoding-experienced-as-retrieval hypothesis.

POSSIBLE MECHANISMS UNDERLYING PATHOLOGICAL DÉJÀ VÉCU

Our initial explanation of these cases is outlined in Moulin et al. (2005). We extended the recollective experience findings from AKP and MA to a tentative neuroscientific mechanism by which inappropriate recollection could be experienced as *déjà vécu*. According to a dual process view (Aggleton & Brown, 1999), there are different structures associated with remembering and familiarity. Aggleton and Brown (1999) proposed that the hippocampus is heavily involved in remembering, whereas the parahippocampal cortex is part of a different system responsible for familiarity. These *déjà vécu* patients were therefore proposed to experience *déjà vécu* characterised by elevated remembering for new items as a result of hippocampal dysfunction (evidenced on structural brain scans of the cases). We propose that the secondary confabulation found to be associated with justifying the sensations of remembering associated with *déjà vécu* was indicative of frontal decline. In short, a hippocampal dysfunction gave rise to sensations of recollection, which due to damage to the frontal lobes (evidenced in neuropsychological test scores) was not corrected, or incorrectly interpreted at a later stage. This combination of hippocampal and frontal networks in the reexperiencing and accepting of memory at retrieval received support from fMRI studies of healthy subjects making “remember” compared to “know” (or familiarity) judgements (e.g., Henson et al., 1999) and from stimulation of the hippocampal areas and associated networks by Chauvel and colleagues (e.g., Bancaud, Brunet-Bourgin, Chauvel, & Halgren, 1994; Vignal, Maillard, McGonigal, & Chauvel, 2007).

Originally, the delusion-like nature of our patients was not explored, but clearly, the role of frontal mechanisms is consistent with the second factor (causing belief evaluation failure) in Langdon and Coltheart’s (2000) model of delusion. A failure in reality testing resulting from frontal decline may result in the maintenance of erroneous recollection in spite of its inconsistency with other aspects of the experient’s beliefs and experiences.

In sum, *déjà vécu* may be a syndrome of dysfunction associated with specific problems related to both hippocampal and frontal decline.

The encoding-experienced-as-retrieval hypothesis

Our earlier account overlooks one key attribute of the *déjà vécu* cases: that the patients tend to experience novel or striking stimuli as having been encountered before. In fact, with these cases it is possible to identify triggers for their experiences, whereas with epilepsy, in contrast, there tends to be no external trigger at all (see Moulin & Chauvel, in press, and O'Connor & Moulin, 2008, for more about familiarity-based *déjà vu* not being triggered by events in the environment). Our strong impression was that whenever attention was engaged by an arousing or novel stimulus, there was an accompanying sensation of *déjà vécu*. We argue that the key feature of these events is that they are striking in that they are novel and that novelty detection is a key step in a process by which attention is orientated towards encoding a stimulus. As an example, when AKP was interviewed for BBC Radio 4, the journalist provocatively asked him if he felt that he had been interviewed before—he had not. AKP described how he had been interviewed before and justified his experience by noting salient perceptual features of his current situation, claiming that it was all exactly as before and using it all as justification of his memory retrieval. In such situations the memory system should be working to *encode* such features for future reference, but AKP misinterpreted such contextual and perceptual information as *retrieval* from memory. In keeping with other authors, we make the assumption that novelty is an important cue for engaging encoding mechanisms, as evidenced by the likelihood that novel events and first-time experiences are remembered for longer periods (Lisman & Grace, 2005; Tulving, Markowitsch, Craik, Habib, & Houle, 1996).

Thus, our more detailed account of the experience of *déjà vécu* is based on the simple principle that encoding-related activity could be misinterpreted as retrieval-related; the rest of this paper concentrates on this proposal. This proposal rests on the idea that the hippocampus supports novelty-detection as well as recollection, and that recollection normally involves a transient and distinct theta-based coupling between the hippocampus and other regions that receive information from the hippocampus. For simplicity, we shall focus our discussion on one output region within the hippocampus, CA1, and one key hippocampal efferent region, the medial prefrontal cortex, but, of course, the same principles are expected to apply to other regions. Note that other authors have suggested a similar disconnection between temporal and frontal regions and resultant confabulation (see Gilboa et al., 2006; Schnider et al., 2002). Broadly speaking, these accounts converge on the orbitofrontal

prefrontal cortex as responsible for gating and monitoring outputs from temporal regions.

Our account assumes that the hippocampus is crucial for reactivating the context associated with an event, and thus for episodic recollection. It also assumes that various neocortical areas (e.g., medial parietal, visual) are involved in recreating the specific contents of the event being recollected. Another assumption is that a third region, such as the medial prefrontal cortex or retrosplenial cortex, is associated with the subjective, conscious experience that these contents evoke, resulting in recollection of one's own past. Medial prefrontal cortex and retrosplenial cortex both receive direct projections from the hippocampus, including CA1 (Amaral & Lavenex, 2007). Using these fairly standard assumptions, we outline a speculative model of how processing might proceed normally, and the consequences of it going wrong. First, we briefly discuss hippocampal involvement in recollection and novelty detection. We then summarise some data and theory on the importance of the theta oscillation in memory. Finally, we suggest a theta-based mechanism of how the encoding of novel experience could be misinterpreted by the brain as the retrieving of that information. We arrive at a specific model of how the interaction between frontal and hippocampal networks may be deficient in such a way as to produce *déjà vécu*. Crucially, this model explains how, in our patients, the act of encoding may be misinterpreted as the act of retrieving.

THE HIPPOCAMPUS AND NOVELTY DETECTION

There is fairly broad consensus that recollection involves the hippocampus (e.g., Duzel, Vargha-Khadem, Heinze, & Mishkin, 2001; Yonelinas, Otten, Shaw, & Rugg, 2005). A lesion study using an animal model of recollection also reached this conclusion (Fortin, Wright, & Eichenbaum, 2004). We emphasise here that several lines of evidence from different research fields demonstrate that the hippocampus also plays an important role in novelty detection (e.g., Honey, Watt, & Good, 1998; Knight, 1996; Kumaran & Maguire, 2007; Lee, Hunsaker, & Kesner, 2005; Nyberg, 2005). It seems likely that this novelty-detection function is directly related to the hippocampal role in episodic encoding of events. First, as the Tulving (1972) quotation beginning this paper implies, any event involves a unique configuration of stimuli to some extent. Second, novelty is graded—very novel, distinct events are preferentially encoded, and endure longer in memory (Lisman & Grace, 2005; Tulving et al., 1996). An episodic memory system should prioritise the recording of experience that is novel and goal-relevant (Lisman & Grace, 2005).

The precise boundaries of the novelty that the hippocampus detects are debatable. Common to most theoretical conceptualisations, however, are that

the hippocampus does not detect simple stimulus novelty but rather a “higher order” novelty. In this higher order novelty, individual stimulus elements may be familiar but the stimulus configuration is novel, or there is a “mismatch” between a stimulus and its context (Honey et al., 1998; Knight, 1996; Kumaran & Maguire, 2007; Lee et al., 2005; Lever, Burton, & O'Keefe, 2006; O'Keefe & Nadel, 1978). A memorable example of potentially hippocampally mediated novelty was provided by O'Keefe and Nadel (1978, p. 241):

“[N]ovelty typically consists of new configurations of familiar elements. . . . [T]he novelty of the wife in the best friend's bed lies neither in the wife, nor the friend, nor the bed, but in the unfamiliar conjunction of the three.”

Clearly, a representation can exist of a new item independent of its context, and this novelty can be detected without the hippocampus. (Such representations occur in perirhinal cortex, e.g., Aggleton & Brown, 1999). An additional novelty engendered by that item is its occurrence in an otherwise familiar, well-mapped context, as in a previously unseen neuropsychologist appearing eagerly at the front door, asking for your participation in a study investigating déjà vu. The hippocampus may well detect this item-in-context novelty (Fyhn, Molden, Hollup, Moser, & Moser, 2002; O'Keefe, 1976) and presumably participate in encoding the event.

Hippocampal novelty detection and the two input streams to CA1

Exactly how the hippocampus detects novelty is not known. Most models, however, assume that region CA1 compares current, real-world, sensory input, which it receives from the entorhinal cortex, with predictive input generated by pattern-completion based recall, which it receives from CA3 (Leutgeb, Leutgeb, Moser, & Moser, 2005; Nakazawa et al., 2002; Wills, Lever, Cacucci, Burgess, & O'Keefe, 2005). When these two input streams do not match, CA1 generates a novelty signal (Hasselmo, Wyble, & Wallenstein, 1996; Kumaran & Maguire, 2007; Lisman & Otmakhova, 2001). Importantly, these two synaptic input streams, one from the entorhinal cortex and the other from CA3, arrive in CA1 at different phases of the theta oscillation. The theta oscillation is an approximately 4–10 Hz oscillation, which is very prominent in the EEG recorded from the hippocampal formation in mammals including humans. Figure 1 shows examples of raw EEG traces recorded from rat and human hippocampus.

Novelty-responsive neuromodulation, including that generated by the hippocampus itself, can change the relative weight of the inputs from entorhinal cortex and CA3, biasing CA1 to be more responsive to its

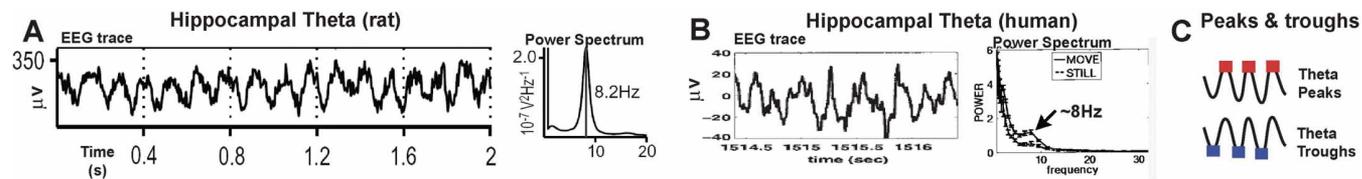


Figure 1. The theta oscillation in rats and humans. (A, B) Hippocampal EEG trace (left) and power spectrum (right) for real exploration (A, rat) and virtual-environment exploration (B, human). Rat power spectrum and human power spectrum during exploration (“MOVE”) show peaks at $\sim 8\text{Hz}$. (C) Schematic theta oscillation showing peaks and troughs (used in Figure 3). (A) is adapted from Jeewajee, Lever, Burton, O’Keefe, and Burgess (2008). (B) is adapted from Ekstrom, Caplan, Ho, Shattuck, Fried, and Kahana (2005). To view this figure in colour, please visit the online version of this Journal.

entorhinal input in novelty (Hasselmo et al., 2002; Meeter, Murre, & Talamini, 2004). Since the input from entorhinal cortex and CA3 arrive at different theta phases, and novelty modulates CA1's response to this input, we might expect CA1 firing during encoding (biased towards entorhinal firing) to show a different mean theta phase from its firing during retrieval (biased towards CA3 firing). To anticipate our argument, a region downstream of CA1 capable of performing theta phase-readout of CA1 activity could underpin judgements about whether the hippocampus is encoding or retrieving.

Background to the new model: Theta, synaptic plasticity, and the separation of encoding and retrieval

Studies in humans and rats strongly indicate the importance of the theta oscillation to memory function (Buzsaki, 2006). Recently, there has been renewed interest in the human theta oscillation following demonstrations that hippocampal theta can be found in humans as well as other mammals (e.g., Kahana, Sekuler, Caplan, Kirschen, & Madsen, 1999; Tesche & Karhu, 2000). Increased theta power or theta coherence is also known to be associated with the success of both encoding and retrieval (e.g., Fell et al., 2003; Guderian & Duzel, 2005; Jacobs, Hwang, Curran, & Kahana, 2006; Klimesch, 1999; Osipova et al., 2006; Sederberg, Kahana, Howard, Donner, & Madsen, 2003). Moreover, theta synchronisation over spatially distributed networks is associated with experiential qualities of consciously remembering (Barbeau et al., 2005; Klimesch et al., 2001), and with retrieval that incorporates correct source judgements (Guderian & Duzel, 2005).

These demonstrations of the importance of theta in human memory have been inspired by work in rats showing strong associations between the temporal signal of the theta oscillation and long-term synaptic plasticity. It is well established, for instance, that long-term potentiation (LTP) in region CA1 of the hippocampus is preferentially induced when stimulation occurs at the peak of the theta oscillation in CA1 (e.g., Holscher, Anwyl, & Rowan, 1997; Huerta & Lisman, 1993). A further finding is that long-term depression (LTD), depotentiation, or no change results if stimulation occurs at the opposite phase to that promoting LTP (Holscher et al., 1997; Hyman, Wyble, Goyal, Rossi, & Hasselmo, 2003). Figure 2A schematically summarises these findings.

In conclusion, a strong determinant of whether information is subsequently remembered or forgotten is the phase of theta that the information arrives at. Consistent with this idea are demonstrations that the brain can optimise memory encoding by scheduling memory-relevant sensory stimuli to arrive at the appropriate theta phase (McCartney et al., 2004; Rizzuto, Madsen, Bromfield, Schulze-Bonhage, & Kahana, 2006; Rizzuto et al., 2003).

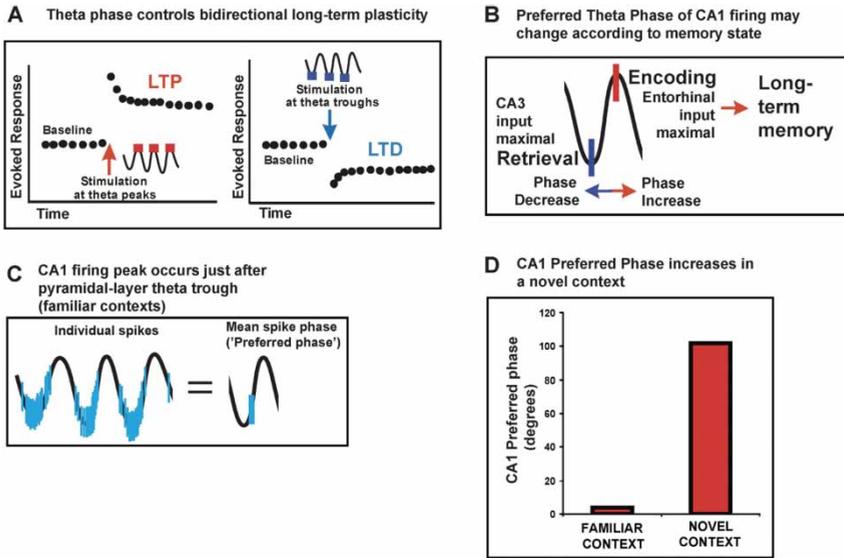


Figure 2. The relationship of theta phase to memory encoding and retrieval. (A) Theta phase controls bidirectional, long-term synaptic plasticity. The phase of theta at which stimulation occurs determines whether hippocampal synapses are subsequently strengthened (long-term potentiation, LTP), unchanged, or weakened (long-term depression, LTD). Schematic summary of data in Hyman et al. (2003) and McCartney, Johnson, Weil, and Givens (2004). (B) The model of Hasselmo, Bodelon, and Wyble (2002) proposes that encoding and retrieval in CA1 typically occur at different phases of theta. This would mean that CA1 preferred phase in novel contexts, when encoding is expected to occur, would be different to that in familiar contexts. The phase of theta when LTP can preferentially be induced in CA1 coincides with maximal entorhinal input, whereas the theta phase when LTD is preferentially induced coincides with maximal CA3 input. (C) Individual spikes of CA1 pyramidal cells occur at various phases of the theta cycle, but it is thought that the “preferred phase” (i.e., the mean phase) of CA1 pyramidal cell firing occurs just after the trough of the local pyramidal-layer theta oscillation (e.g., Csicsvari, Hirase, Czurko, Mamiya, & Buzsaki, 1999). This result was based on testing in familiar environments: Novelty variables were not studied. (D) In a novel context the preferred theta phase of firing in CA1 is increased relative to preferred phase in a familiar context. Mean all-spikes theta phase shown for one rat (two trials in familiar context, one trial in novel context). Mean theta phase is normalised such that the last trial in the familiar context is 0° (Lever et al., unpublished data). To view this figure in colour, please visit the online version of this Journal.

On the basis of such data, it has been proposed that the theta oscillation separates encoding and retrieval modes in the hippocampal memory system (Hasselmo et al., 2002; Hyman et al., 2003; Judge & Hasselmo, 2004; Kunec, Hasselmo, & Kopell, 2005). A basic prediction of these models is that the mean phase of firing across the CA1 population will differ according to whether CA1 is encoding novel information (entorhinal biased) or retrieving old information (CA3 biased). See Figure 2B for a schematic diagram of this model. Previous studies have investigated the mean phase (also called

“preferred phase”) of CA1 firing in rats, and found that the mean phase occurs just after the theta trough in the pyramidal layer (e.g., Csicsvari et al., 1999). See Figure 2C for a schematic summary of these findings. These studies were based on rats foraging in familiar environments. Recent work has explicitly looked at theta phase of firing during manipulations of novelty. This recent work (Lever et al., unpublished data; Manns, Zilli, Ong, Hasselmo, & Eichenbaum, 2007) supports the view that theta phase separates encoding and retrieval states. Lever et al. (unpublished data) consistently showed an *increase* in mean theta phase associated with novelty; that is, spikes of CA1 pyramidal cells tended to fire *later* in the theta cycle in a novel, unexpected environment than in an expected, familiar environment (Figure 2D).

The new model: Mean theta phase and an erroneous signal of “retrieval”

In summary, recent evidence indicates that the principal cells in CA1 (the output region of the hippocampus proper) fire at different mean phases of the theta oscillation depending on whether they are primarily encoding or primarily retrieving information. To our knowledge, in the work reviewed above Hasselmo and colleagues have not emphasised the potential consequences of the encoding versus retrieval theta phase difference for regions *downstream* of the hippocampus. If we assume that the same CA1 neurons participate in both encoding and retrieval, memory state (i.e., encoding or retrieval) cannot be inferred on the basis of which subset of CA1 neurons fire. Indeed, the more accurate the memory, the greater the overlap will be between the set of neurons that fired during the original experience and the reactivated neurons, whose firing subsequently helps to recreate, and thus subserve the remembering of that experience. What should be different about the original encoding and the subsequent retrieval is the average theta phase of CA1 spiking. Thus, it seems plausible to suggest that phase readout (e.g., Jensen, 2001) in downstream regions could play an important role in interpreting whether hippocampal output represents memory encoding or retrieval. Strength of activation per se may not be a reliable signal of hippocampal memory state.

Our proposal, then, is simply that downstream regions (e.g., medial prefrontal or retrosplenial cortex) which receive direct projections from CA1 detect this encoding versus retrieval theta phase difference. There is emerging evidence for medial prefrontal neuronal firing being phase-locked to the hippocampal theta oscillation when this is appropriate to the current behavioural task (Jones & Wilson, 2005a,b; Siapas, Lubenov, & Wilson, 2005). Interestingly, during the part of a spatial memory task when the medial prefrontal cortex most needs to read hippocampal output, theta coherence transiently increases, and the population activity of medial prefrontal neurons

becomes more phase locked, indeed converging to a particular phase of the hippocampal theta rhythm that is consistent across different rats (Jones & Wilson, 2005a). The convergence to a *particular* phase of the hippocampal theta rhythm could mean that prefrontal firing at *that* particular hippocampal theta phase is associated with, and is effectively the signature of, prefrontal interpretation of hippocampal *retrieval*, thus “recollection”. One might also imagine prefrontal neurons further downstream which only fire when there is sufficient input from CA1 *and* when this input arrives at the appropriate phase of theta. Interestingly, if this is the case, we would arrive at a mechanism by which a set of neurons typically only fire when there is strong theta coupling between the hippocampus and prefrontal cortex, *and* when the hippocampus is in retrieval mode. The firing of this further-downstream set of neurons would in effect signal recollection. Although these neurons in the normal situation would be under tight inhibitory control and would only fire under theta-coupling states, this coding scenario would allow for the possibility that “recollection” neurons could be activated pathologically, thus eliciting a false signal of recollection, as occurs in *déjà vécu*.

ENCODING STATE INTERPRETED AS RETRIEVAL STATE IN DÉJÀ VÉCU

Our account, then, assumes three states for the hypothesised downstream region (e.g., medial prefrontal cortex) with respect to hippocampal output:

1. Not receiving (no theta coupling);
2. Receiving (increased theta coupling) while hippocampus is encoding (theta encoding phase); and
3. Receiving (increased theta coupling) while hippocampus is retrieving (theta retrieval phase).

Our contention is that in pathological *déjà vécu*, there is a disruption to the timing of firing relative to the theta oscillation. This disruption is such that the phase of firing of prefrontal neurons is that associated with hippocampal retrieval, even though the CA1 neurons contributing to the excitation of the prefrontal neurons may actually be firing in a novelty-responsive mode supporting encoding. The precise reason why a timing disruption might occur is unclear and could result from disruption in prefrontal cortex of the integration of hippocampal synaptic input, disruption to hippocampal or prefrontal spike timings relative to a normally functioning theta oscillation, impairments in phase reset of ongoing theta, or impaired synchronisation between septohippocampal and neocortical theta. Although it is anticipated that hippocampal dysfunction plays a role in this timing disruption, what we

predict is *unimpaired* in déjà vécu is the hippocampal novelty detection mechanism.

Although our hypothesis is not testable using fMRI, it can be tested using electrophysiological recordings of both EEG and single neuron firing patterns. Using electrodes implanted in the hippocampus, evidence in support of our hypothesis would include the following:

- When stimuli are presented that *elicit false recollection*, CA1 or the downstream neocortical region neurons fire with a *similar* mean theta phase of firing as that occurring during genuine recollection of past events.
- However, when stimuli are presented that *do not elicit false recollection*, the CA1 or neocortical neurons fire with a *different* mean theta phase of firing to that occurring during genuine recollection.

A complementary approach is to show that the downstream neocortical neurons, which typically fire during recollection-elicited states of high theta coupling between the hippocampus and the neocortical region in question, can also fire in abnormal circumstances (seizures, pharmacological intervention), and when they do fire, subjective sensations of “recollection” accompany such firing.

Finally, we return to the scenario of one of our déjà vécu patients making a characteristic error. Consider the act of making a false positive response, i.e., stating that an item was present at the presentation phase of the test, accompanied by the feeling of “remembering” this item. Such an error, is by definition only made for nonpresented (i.e., *novel* in that context) items. Following the first presentation of a contextually novel word, *Christmas*, hippocampal mechanisms signal that the stimulus is contextually novel. Resources are directed towards this novelty, and neuronal firing in the hippocampus attempts to encode this word and associated background features. The patient (like all of us) does not have direct conscious access to whether this hippocampal activity represents encoding or retrieving. This judgement is neocortically mediated, by a region to which the hippocampus projects, and that reads out the preferred theta phase of CA1 firing. The phase readout process is impaired such that hippocampal CA1 activity arrives at a theta phase associated with retrieval. As a result, the first presentation of a novel word, *Christmas*, results in the sensation of “recollection”. The authenticity of this sensation inspires two kinds of post hoc rationalisation searching for potential causes leading to the sensation. First, stimuli in the current sensory environment are attributed the status of “having-already-been-experienced”. Second, confabulations are generated, whereby actions are

inserted into the narrative of the patient's past to explain just how the patient could actually have experienced the novel event.

CONCLUDING REMARKS

In this paper we have attempted to (1) outline differences between false recognition made with the sensation of recollection and familiarity, suggesting that false recognition on the basis of familiarity might be termed *déjà vu* and on the basis of recollection, *déjà vécu*, and (2) elucidate a neuroscientific mechanism by which the ironic novelty errors occur in our cases of *déjà vécu*. To advance our understanding of familiarity and recollection processes, we suggest that patients' false recollections be classified according to Distinction 1, in the hope that we might be able to further distinguish between the different types of memory errors that the patients make. In terms of the neuroscientific aspects of our model, we have arrived at an account that emphasises the importance of theta phase readout and theta coupling between the hippocampus and regions downstream of the hippocampus. In doing so, we have refined our original frontotemporal account of *déjà vécu*. Continuing to investigate similar cases with particular emphasis on responses to novelty could help to further elucidate the hypothesised, theta-mediated relationship between novelty detection and false recollection.

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